Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services
Emergency Programs

Number 12-2 June 1984

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Current Events

Avian Influenza Update

A national animal disease emergency for avian influenza (AI) in the States of Pa., Va., N.J., and Md. was reported in the March 1984 issue of the Foreign Animal Disease Report (12-1).

Since the start of the calendar year the number of premises with AI in the quarantined area of Pa. increased by 80, bringing the total to 289 premises. The rate of new occurrences decreased from an average of five per day in January to less than five per week in March, with no new cases reported during the first 10 days of April.

A portion of Franklin County, Pa., that was within the quarantine area was released from quarantine on March 1. Also, on March 6, the 12-square mile area that had been under quarantine in N.J. was released from quarantine. New Jersey is the first State to be released from quarantine in the current outbreak. The quarantined area of Md. was released April 5, 1984, making it the second State to have all quarantines released. A total of 203 premises have been released from quarantine in the eradication campaign.

Repopulation has started in the quarantined area of Pa. Seventy flocks of laying hens, 55 flocks of broilers, 17 flocks of pullets, and a flock of breeding chickens have been established on cleaned and disinfected premises.

In Va., the AI Task Force has declared 61 flocks infected with avian influenza; 58 have been depopulated. Most of the affected poultry in Va. are turkeys.

A poultry surveillance program for AI antibodies and virus is in full operation in Pa. and Va. Separate surveillance operations are in progress in W. Va., adjacent to the quarantined area of Va. (Dr. Allan A. Furr, 301 436-8091).

Avian Influenza in California

A primary turkey breeder flock, located at Waterford, Calif., was found to be infected with AI virus type A, subtype H5N3, during the week ending March 10, 1984. This isolate failed to kill any of the test chickens inoculated in the laboratory. There was no connection with the outbreak in Pa., which was caused by a different virus subtype, H5N2. The flock was humanely slaughtered and buried on the premises March 11. Costs of depopulation and disposal were paid by the California Poultry Industry. Cleaning and disinfection of the premises were conducted under supervision of the State-Federal Emergency Disease Organization. A second infected flock was depopulated March 17. Four other premises are under observation. (Dr. Allan A. Furr, 301 436-8091)

Wildlife Studies on Avian Influenza

A major epidemiologic question in the eruption of lethal AI in Pa. during 1983 was the possible role of wildlife in its introduction and spread. Authorities on influenza viruses generally consider wild birds, particularly waterfowl, as asymptomatic reservoir hosts for a wide variety of AI subtypes and strains. Therefore, a wildlife section was immediately established in the AI Task Force to assess the potential for wild birds to spread disease locally among farms or carry the virus to distant areas.

Through a cooperative agreement with Veterinary Services (VS), the Southeastern Cooperative Wildlife Disease Study (SCWDS) of the University of Ga. led the wildlife section's search for subtype H5N2 AI virus and antibody in wild birds and small rodents. Laboratory support for the survey was provided by the World Health Organization's (WHO) Collaborating Center for Studies on the Ecology of Influenza in Animals, at Memphis, Tenn.

Priorities were placed on sampling wild ducks, free-flying domestic ducks, geese, and seagulls; wild birds closely associated with poultry farms, poultry manure, or carcasses; mice and rats found near infected poultry houses; and wild birds reported sick or dead within the quarantined area. Wildlife specimens were obtained by a variety of methods, including donation by hunters, trapping, netting, and shooting by SCWDS biologists. Wherever feasible, animals were collected on farms where AI was present. All reports of sick or dead wild birds were evaluated for possible influenza infection.

Results of over 4,300 virus isolation attempts (table 1) indicated that if indeed AI subtype H5N2 was present in wildlife, it could have been present only at an extremely low prevalence. An H5N2 virus was isolated from one pheasant submitted to the National Veterinary Services Laboratories, at Ames, Iowa, early in the eradication effort. Studies at St. Jude Children's Research Hospital indicated that pheasants could be infected experimentally. Nevertheless, this species is not very abundant around poultry farms, and 58 other wild pheasants have tested negative. A pen-reared chukar maintained on a poultry premise which became infected also was found to be infected with H5N2.

Table 1--Numbers of birds and rodents collected in the quarantined zone of Pa. and results of tests for avian influenza virus A, subtype H5N2

	Total specimens collected	Total isolations of virus	Total specimens without virus	Total specimens not tested to date
Species	Number			
Wild Ducks	537	0	534	3
Free Ranging Domestic Ducks	208	0	208	8
Wild Geese	512	0	504	8
Free Ranging Domestic Geese	16	0	16	0
Wild Swans	13	0	10	3
Seagulls	208	0	205	3
Crows	201	0	198	3
Starlings	569	0	569	1
Starling Feet	16	0	16	0
Red-Wing Blackbirds	14	0	14	0
Cowbirds	72	0	72	0
Sparrows	546	0	544	2
Sparrows Feet	178	0	171	7
Pigeons	473	0	458	15
Wild Pheasants	69	1	58	10
Pen-reared Pheasants	198	0	138	60
Pen-reared Chukars	71	1	41	29
Pen-reared Quail	76	0	30	46
Vultures	17	0	4	13
Miscellaneous Birds	34	0	26	8
Mice	245	0	233	12
Mice Feet	223	0	208	15
Rats	24	0	24	0
Rat Feet	24	0	24	0

Cloacal and tracheal swabs from aquatic birds, that is, ducks, geese, and seagulls, have yielded four influenza viruses different from the H5N2 virus, and 11 paramyxoviruses. It is of interest to note that one seagull had an influenza virus that did not kill embryonated eggs. The low frequency of influenza virus isolation is comparable to the results of other studies of water birds during winter months.

While wildlife studies were progressing in the quarantine zone, SCWS obtained samples from 1,000 wild geese and 500 ducks on the Chesapeake Bay. A total of 645 of these birds have been tested without evidence of H5N2 AI virus.

Studies completed have not revealed the source of the H5N2 virus that caused the outbreak of disease in domestic poultry. Results of tests for antibodies indicate only water birds were infected with H5 influenza viruses in the recent past (table 2).

Table 2--Numbers of bird and rodent serums collected in the quarantined zone of Pa., and results of tests for type 5 hemagglutination inhibiting antibodies (H5), and type 2 neuraminidase-inhibiting antibodies (N2)

	Total Serums Tested	Total serums reacting (+) or failing to react (-)			
		H5- and N2-	H5+ but N2-	H5- but N2+	H5+ and N2+
Species	Number				
Wild Ducks	385	258	68	30	29
Domestic Ducks	42	24	12	1	5
Wild Geese	130	100	11	13	6
Domestic Geese	10	9	0	0	1
Seagulls	156	102	26	15	13
Crows	127	127	0	0	0
Starlings	479	479	0	0	0
Red-Wing Blackbirds	8	8	0	0	0
Cowbirds	34	34	0	0	0
Sparrows	522	522	0	0	0
Pigeons	361	361	0	0	0
Wild Pheasants	4	4	0	0	0
Pen-reared Pheasants	26	26	0	0	0
Mice	103	103	0	0	0
Rats	23	23	0	0	0

Results of these studies suggest that wildlife such as ducks, geese, seagulls, crows, blackbirds, starlings, sparrows, pigeons, mice, and rats did not play a significant role in the spread of AI virus among farms within the quarantine zone. Gallinaceous game birds, such as pheasants and chukars, were shown to be susceptible to H5N2 influenza virus. Therefore, they could be considered potential spreaders of the virus, especially on game bird farms where bird densities are high.

Although aquatic birds had influenza antibodies suggesting prior infection with viruses of the H5 subtype, their possible role in the initial outbreak in Pa. is uncertain. Influenza viruses are known to circulate through free-flying aquatic birds during the spring and early summer months, when a large number of juvenile birds enter the bird population. Additional surveillance should be conducted on aquatic birds during the spring and summer of 1984 to obtain valuable information about the current and future status of H5 viruses in migrating species. (Dr. V. F. Nettles, 404 542-1741; Drs. J. Wood and R. G. Webster, 901 522-0400)

Focus on... Avian Influenza

Avian influenza (AI) is a disease of viral etiology that ranges from an asymptomatic infection to an acute, fatal disease of chickens, turkeys, guinea fowl, and many other avian species.

The Virus

The AI viruses, along with the other mammalian influenza viruses, belong to the family Orthomyxoviridae. Animal influenzas, including AI and most human influenza infections, are caused by type A influenza viruses.

The designation of viral type is based upon internal antigens, which are readily demonstrated by immunodiffusion and complement fixation tests. The AI viruses, like the other influenza viruses, have surface projections or spikes 10 to 12 nanometers long. These projections are the hemagglutinin (H) and neuraminidase (N) antigens. Both the H and N antigens are important in identifying subtypes of the influenza viruses using inhibition tests (HI and NI). There are 13 hemagglutinin and 9 neuraminidase antigens among the many AI viruses which determine their subtype designation.

The genetic material (RNA) of AI virus is divided into eight distinct segments, a characteristic that is important in the evolvement of new viruses through genetic reassortment in hosts that are infected with more than one influenza virus at the same time.

What is Fowl Plague?

Fowl plague (FP) is one of the most notorious members of the AI viruses. In 1955, FP was determined to be one of the AI viruses with H7 (formerly Hav 1) antigen and any of several N antigens. Investigators initially believed that all H7 AI viruses were FP and highly lethal. But in 1971, an AI virus with H7 was recovered in Oregon from turkeys with very mild disease. It was soon apparent that there could be both mild and virulent avian viruses of each H designation and that disease clinically indistinguishable from FP could be caused by non-H7 avian viruses. The disassociation of AI virulence from a particular H antigen, as evidenced by the finding of numerous highly pathogenic AI viruses with different surface antigens, resulted in a recommendation adopted by the First International Symposium on Avian Influenza in Beltsville, Md., in 1981. This recommendation was to use the term "highly pathogenic avian influenza" (HPAI) as a substitute for the H7 specific term of fowl plague. HPAI was to apply to those AI viruses that would result in the death of at least 75 percent of a total of eight chickens, 4 to 8 weeks of age, within 8 days after they were inoculated with the virus by intramuscular, intravenous, or caudal air sac routes.

AI Onset and Signs

The incubation period of virulent AI in laboratory chickens is usually 3 to 4 days. In field situations, where the first affected bird may not be detected and the infecting dose is low, the incubation period may be 5 to 7 days. This can vary, however, depending on the virus, host species, and conditions of exposure.

Many unique features characterize the AI viruses. For example, the severity of clinical disease ranges from no obvious signs to clinical FP-like disease with mortality rates approaching 100 percent. The type of disease also varies. Some strains of the virus induce severe respiratory signs with or without diarrhea. Other isolates produce little respiratory involvement but severe diarrhea. Birds affected with a highly virulent virus may sicken and die without significant disease signs other than severe depression. The age of the chicken can markedly influence the clinical picture and mortality rate following experimental inoculation. Adding another variable, a particular virus may produce severe disease in one avian species but not in another.

The fowl plague-like disease that results from highly pathogenic AI infection can be striking in chickens and turkeys, with initial signs of inappetence, severe depression, diarrhea, and a marked decline in egg production. The last eggs laid can be frequently without shell color in turkeys and are soft shelled in both species. The involvement of the swollen and cyanotic combs and wattles may be so extensive as to produce vesicles filled with clear or blood-tinged fluid. Some chickens may exhibit marked, diffuse congestion of the shanks.

Turkey flocks with AI may display less severe signs of disease than chicken flocks affected by the same strain of virus. Turkeys may also exhibit a greater incidence of central nervous system signs than do chickens.

Respiratory disease and sinusitis have been associated with some AI infections in ducks, but in many instances there has been no apparent disease in this species.

One of the features most frustrating when attempting to characterize AI has been the inability of scientists to take virus from a flock with 30 to 50 percent mortality into laboratory chickens or turkeys and produce similar mortality rates. Even with highly virulent AI viruses that produce FP-like disease, the laboratory-induced disease may be quite variable, depending upon the age of the inoculated chicken and, in some instances, undetermined influences.

History

The avian influenza problem was quiet in the United States between the last FP occurrence in N.J. in 1929 and that of the early 1960's. It then began to appear on a rather regular basis in turkey flocks in many parts of the country, often resulting in mild disease with declines in egg production. Some flocks experienced more severe losses, and in the winter of 1978-79, Minnesota turkey flocks suffered losses from AI estimated at $5 million.

Between 1929 and the current AI outbreak, the problem in chickens was confined to two small outbreaks. One was in three laying flocks in Alabama in 1975, where the mortality rate declined dramatically from the first affected flock to the last. The other affected premises, in Minnesota in 1979, involved three flocks of layers in connected houses with low and variable levels of mortality. These two chicken outbreaks were caused by H5N8 and H6N1 viruses, respectively. They appeared to be self-limiting because the disease did not spread to other chicken flocks.

1983 Outbreak

The AI outbreak that has received so much attention recently began in southeast Pennsylvania in the spring of 1983. It appeared in chickens, both broilers and layers, and resulted in some mortality. Death was frequently due to caseous plugs in the trachea. Fowl plague-like signs were not characteristic of the early disease, but began to appear in October. The more severe disease was clinically indistinguishable from the classical, textbook descriptions of fowl plague. The milder viruses that first appeared in the spring also continued to circulate in the area. The viruses causing both types of disease were serologically indistinguishable and were identified as subtype H5N2.

The appearance of highly virulent virus in the fall of 1983 led to State, Federal, and industry efforts to eradicate AI in Pa. and Va. Single flock extensions into N.J. and Md. were successfully eradicated and quarantines lifted. The Va. viruses did not produced as severe a disease in poultry flocks as that observed in Pa. Nevertheless, through epidemological investigations, the Va. outbreak was shown to be related to the Pa. outbreak.

Diagnosis

Since the signs of disease caused by AI are variable, diagnosis can be complicated. It usually must await isolation and identification of the virus. In an area where a highly lethal AI virus is known to exist, a presumptive diagnosis can be made on clinical signs and post-mortem lesions. The diagnosis may then be confirmed by laboratory tests demonstrating the virus and/or specific antibodies.

On post-mortem examination, it is not unusual to observe yolk peritonitis, hemorrhages on the ovary, and fine spray paint petechiae on the abdominal fat, pericardium, and underneath the keel. There may be hemorrhages in the proventriculus and under the readily peeled lining of the gizzard. The pancreas is occasionally grossly affected. Kidneys may be swollen or filled with deposits of urates. When AI occurs in a new area, it is difficult if not impossible to make a firm distinction between

virulent AI and viscerotropic velogenic Newcastle disease, based on clinical signs and gross post-mortem lesions.

AI virus is shed in respiratory secretions and feces. AI viruses have also been recovered from the yolk and albumen of eggs laid at the onset of severe disease. Al viruses are frequently isolated from soft-shelled eggs laid by infected chickens. The AI viruses are readily recovered by inoculating the chorioallantoic sac of 9- to 10-day-old embryonating eggs. After 48-72 hours of incubation, the embryo may die and virus may be demonstrated in the chorioallantoic fluid by the hemagglutination (HA) test.

The inhibition of HA activity by monospecific antisera against known H antigens is diagnostic. Since there are 13 known H antigens to be tested for, by use of the hemagglutination-inhibition (HI) test, the agar gel diffusion precipitin (AGP) test is often used. The AGP test detects internal antigens shared by all Type A influenza viruses, regardless of their H designation.

When HA activity is demonstrated in inoculated eggs and that activity is not inhibited by specific Newcastle antiserum, the chorioallantoic membranes can be removed, homogenized, and used as an antigen in an AGP test against a known Type A influenza antiserum. Once an AI virus with a known H antigen is established as the causative agent in a disease outbreak area, diagnosis can be simplified by applying the specific HI test.

Poultry and other birds with AI develop antibodies very rapidly. About half of the chickens experimentally infected with several of the Pa. isolates had positive sera when tested with the AGP test 5 or 6 days later. That means that half of the chickens were AGP-positive as early as 2 days after the first signs of disease. The AGP test will give positive results following infection with any influenza A virus. The HI test can then be applied to identify the specific H type.

Prevalence and Hosts

Avian influenza is worldwide. In the United States, outbreaks have usually resulted in mild disease, most frequently in turkeys, and have generally been self-limiting. However, AI infections have sometimes caused severe mortality in poultry flocks. Australia, England, Canada, Scotland, Ireland, and the United States have had localized outbreaks of highly virulent AI. Numerous other countries have reported problems of variable severity in a variety of avian species. The U.S.S.R., Italy, Israel, France, Belgium, and Hong Kong have experienced AI infections of domestic avian species in recent years. Some of the highly virulent viruses had the H7 surface antigen, while others had H5. The H7 and H5 surface antigens are frequently associated with severe disease, even though there is no direct relationship between the identity of surface antigens and virulence. Some H5 and H7 viruses are relatively mild and cause minimal disease.

The AI problem that has been recognized since the mid-1960's as a poultry disease problem has been rare in chickens. Scotland experienced a virulent H5-induced chicken disease in 1959.

Reports from the U.S.S.R. indicate that chickens have frequently experienced severe losses to AI since the mid-1960's. With the exception of the U.S.S.R., the isolated flock outbreaks in Australia, and two occurrences in the United States, AI has been primarily a problem of turkeys. The current Pa. outbreak has dramatically altered that host-associated conception of AI because both chickens and turkeys have suffered severe morbidity and mortality.

Wild ducks and sea birds are frequent sources of AI viruses in cloacal swab surveys. Generally, the viruses are recovered from young, apparently healthy ducks, usually in the fall, as they leave the nesting grounds for warmer climates. There is a considerable body of circumstantial evidence to indicate that these avian species serve to distribute the viruses and directly or indirectly introduce them into poultry flocks.

Studies performed since the early 1950's on domestic ducks have yielded a variety of AI viruses with different H antigens. Infected but normal appearing domestic duck flocks have been incriminated as sources of virus that caused high mortality in nearby chicken flocks.

The AI viruses occasionally cause severe death losses in wild birds, as evidenced by the isolation of an H5 AI virus from a die-off of common terns in South Africa. Numerous other species of sea birds have been the source of AI viruses in the absence of obvious disease.

Disease Control

There has been considerable debate on the appropriate means to control AI. Some have proposed that the proper way to deal with the recent Pa. outbreak of AI is to apply the same methods used with previous outbreaks of less pathogenic AI viruses in turkeys. Past control measures in turkeys with AI viruses that did not kill laboratory chickens included isolation of infected flocks, use of killed vaccine in surrounding flocks, and marketing of infected flocks through normal channels after recovery.

Several factors that make this approach a potentially costly one include trade embargoes, vaccination costs, and spread of virus to additional flocks. The occurrence of FP-like AI virus in Pa. has resulted in disruptive embargoes by some countries, even though the affected areas are under Federal-State quarantine. Also, other U.S. poultry growers not wishing to bring the diseases into their areas, have implemented many practices to lessen the chances for such an occurrence. Both of these consequences can be prolonged and become more widespread with a program of vaccination and marketing of infected but normal-appearing poultry.

Measures used to control AI viruses that have occurred in the U.S. prior to the current outbreak are considered inadequate to assure eradication of highly pathogenic AI. Cooperative eradication efforts currently in use by the poultry industry and State and Federal animal disease control agencies consist of surveillance to detect infected flocks, depopulating and deep burial of the carcasses or disposal in approved landfills, and cleaning and disinfecting the premises.

A complication of the Pa. eradication program resulted from the simultaneous circulation of both mild and highly pathogenic AI viruses in affected poultry flocks. The mild virus appeared to alter the kind of disease produced when both mild and virulent viruses occurred in the same flock. To expedite eradication, a decision was made to eradicate all flocks in the quarantine area infected with serotype H5 regardless of the disease signs in the flock or the disease-producing capacity of the isolate in laboratory chickens.

The use of vaccine was not included as part of the eradication effort because it could greatly reduce the severity of disease signs in flocks exposed to AI, thereby delaying the detection of virus-shedding flocks. Vaccine can cause antibodies that would reduce or negate the value of serology in determining the AI infection history of flocks in an area within or near the quarantine zone. Testing flocks at slaughter plants for AI antibody and testing for AI antibody in egg yolks are excellent ways to search for AI activity in the area supplying the samples. However, the test is only useful in areas where no vaccine-produced antibodies are present. Also, due to a required 42-day interval between the use of oil-emulsion vaccine and the slaughter of broilers, the use of vaccine in that segment of the industry is impractical.

There is no doubt that vaccine can result in reduced losses in individual poultry flocks. Its use may also be an important tool in controlling the less severe AI viruses. But, the concensus now is that vaccine should not be used as part of an attempt to eradicate AI when highly virulent or lethal strains of the virus are present.

A basic problem with vaccination for influenza control results from the large number of viruses with different H antigens that can cause disease. To have an inventory of vaccines that would be effective against any of the known viruses that have infected poultry would require at least 13 vaccines or multivalent vaccines that contained representatives of the 13 antigens. There have been years when as many as four H antigens were represented in AI viruses causing losses in one State.

Looking Ahead

The current outbreak has brought new attention to the importance of sanitation and security for prevention and control of all diseases, including AI. The remote location of new housing, careful entry of equipment and personnel into flocks, proper disposal of dead birds, control of rodents and insects, and improved disease reporting systems will aid in preventing AI and reducing losses.

The events of 1983 have also clearly shown that any appearance of AI in chickens in the future should be viewed with great caution, and immediate steps should be taken to contain and eliminate it.

Because free-flying waterfowl and sea birds are the suspected source of periodic outbreaks of AI in domestic poultry, future efforts to prevent the disease must be directed at strict

isolation of domestic poultry from direct or indirect contacts with wild bird hosts. No one can accurately predict when or where new introductions may occur, or which of the 13 known hemagglutinins will be represented. If past records can be a basis for predictions, the industry may operate for many more years without another FP-like outbreak like the one that occurred in Pa. The industry must make improved sanitation and security a part of its overall and continuing management practice, and not just a procedure implemented in the face of impending disease.

The apparent transition by mutation of a mild virus in Pa. to a highly pathogenic virus producing an FP-like disease, after circulating in chicken flocks for 6 months, gives cause for caution in discounting the potential of other avirulent AI viruses to become highly pathogenic. The need for research to achieve a better understanding of the mechanisms of AI virulence and epidemiology is readily apparent from the recent and current experiences. Because of the widespread nature of the viruses in free-flying species, this disease may present continuing and unprecedented challenges to the poultry industry and disease control specialists. (Dr. Charles W. Beard, Director, Southeast Poultry Research Laboratory, ARS, USDA, Athens, Ga., 404 546-3432).

More Current Events

Vesicular Stomatitis in Texas

Vesicular stomatitis was diagnosed in cattle on three premises in central Texas in early February. New Jersey-Type vesicular stomatitis virus was isolated from tissue from one steer. This outbreak was associated with a group of roping steers imported from Mexico in early January. History obtained at the time of diagnosis included a report of clinical signs and typical lesions of vesicular disease in some of these cattle in mid-January. Most of the lesions were healed when the affected animals were first observed by foreign animal disease diagnosticians, leaving slight opportunity to obtain tissue samples. (Dr. Allan A. Furr, 301 436-8091).

Puerto Rico Tick Program

The special report on the Puerto Rico tick program in the December 1983 issue (11-4) contained misleading information. The following correct information replaces corresponding data given in the earlier article.

Except for the periods 1941-46 and 1968-78, Puerto Rico has been under quarantine for ticks since 1936. The most recent quarantine was extablished in 1978, following the identification of southern cattle ticks, Boophilus microplus, on the island. The tropical bont tick, Amblyomma variegatum, was found in Puerto Rico in May 1974, and its identity was confirmed a month later. This finding did not mandate a quarantine in 1974 because the applicable Federal regulation--9 CFR 72--specifies quarantines for only three tick species: B. annulatus, B. microplus, and Rhipicephalus evertsi evertsi. Title 9, Code of Federal Regulations, Part 72.3, states that the entire territories of the Virgin Islands of the United States, the Island of Guam, the northern Mariana Islands, and the Commonwealth of Puerto Rico are quarantined. Part 72.5

describes the area quarantined in Texas. (Dr. Bob H. Bokma, San Juan, Puerto Rico, 809 724-0466).

World Animal Disease Roundup

Occasionally one hears debate on the exotic disease causing the most problems worldwide. The disease most often incriminated is **foot-and-mouth disease** (FMD). An outbreak of FMD in the Netherlands, reported in the March 1984 issue (12-1), spread to a total of six premises before it was contained. The last case occurred February 2, 1984. Elsewhere, Chile had a disappointing experience. The United States recently added it to the list of countries considered free of FMD--the first country in South America to achieve that status--only to remove it again on March 29, 1984, when FMD returned to Chile in diseased cattle smuggled from Argentina (OIE Telex dated March 23, 1984). While it is too early to make a final judgment, it appears that the cattle were intercepted before there was a chance of exposing large numbers of domestic Chilean livestock.

Denmark was returned to the list of countries considered free of FMD on January 16, 1984. Denmark last experienced FMD in a herd of cattle on the Island of Funen on January 13, 1983, as reported in the June 1983 issue (11-2).

Contagious bovine pleuropneumonia (CBP) was found again in southern France just as problems with this disease were considered eliminated. CBP was last reported in France 2 years ago. Infected cattle apparently acquired the disease while on summer pasture with livestock from Spain.

Mali reported new cases of **rinderpest**, giving Veterinary Services personnel stationed there an opportunity to observe the disease firsthand. The devastating rinderpest epidemic now going on in Africa was described in the December 1 issue (11-4). Saudi Arabia continues to have problems with the disease in livestock imported on-the-hoof for slaughter. (Dr. H. J. Seyffert, 301 436-8285).

Errata

In the article on World Animal Disease Roundup, December 1983 issue (11-4), the first paragraph should have read, "Glanders was reported only in Turkey." This should have been followed by a paragraph stating, "Dourine, regularly reported from South Africa and Namibia, also resists eradication efforts in Italy, where a few cases have been reported almost monthly." According to official documentation from Namibia, glanders was eradicated in that country in 1925. (Editor, FAD Report, 301 436-8087).

Training

A training course on emergency animal disease eradication for military support veterinarians was held April 16 to 20, 1984, at Hyattsville, Md.

A training course for foreign animal disease diagnosticians is scheduled September 11 to October 2, 1984. The course will begin at Ames, Iowa, continue at Plum Island Animal Disease Center, and end at Hyattsville, Md. Trainees were nominated by Veterinary Services area and regional officials and others. There were approximately four candidates for each training position. (Dr. Allan A. Furr, 301 436-8091).

BAI Centennial

The May 29 commemoration ceremonies for the Bureau of Animal Industry (BAI), announced in the March issue (12-1), came off in grand style. Around 250 people attended. Embassies of 30 foreign countries were represented. Seven Deans of Veterinary Schools and four State veterinarians were present. Representation from farm, livestock and humane groups read like a "who's who in agriculture." The seminar presentations looking at the next century of animal health were stimulating and well presented. The exhibits and animals attracted much favorable attention. (APHIS News Center, 301 436-7799).

100 Years of Animal Health

The 100th anniversary of the first nationally organized efforts to control and stamp out dangerous animal diseases occurs in 1984.

On May 29, 1884, Congress created the Bureau of Animal Industry (BAI) within the U.S. Department of Agriculture to "prevent the exportation of diseased cattle and to provide the means for the suppression and extirpation of pleuropneumonia and other contagious diseases among domestic animals."

Celebration of this anniversary is more than a bureaucratic milestone. Proper nourishment of millions of people depends on the vital animal protein supplied by healthy livestock and poultry.

Today our huge and complex livestock industry owes its remarkable growth and productivity, in part, to the cooperative efforts of various government agencies working with producers to identify, control, and wipe out costly and dangerous animal diseases. Diseases and arthropod pests eradicated from the United States include foot-and-mouth disease, fowl plague, dourine and glanders in horses, cattle fever ticks, screwworms, vesicular exanthema of swine, Venezuelan equine encephalomyelitis, sheep scabies, exotic Newcastle disease in poultry, and--most recently--hog cholera.

There have been significant spinoffs from the work of the Bureau and its successors. In the late 1800's, three BAI scientists proved that ticks spread Texas fever in cattle. This discovery in the field of animal health led to control of yellow fever in humans and enabled U.S. workers to complete the Panama Canal.

In 1905, novelist Upton Sinclair published "The Jungle," a scathing expose of filthy conditions in packing plants. Public opinion soon mobilized legislation: The Meat Inspection Act of 1906. Today's consumers buy with confidence, secure in the knowledge that when USDA's inspection stamp goes on a meat or poultry product, it is safe, wholesome, and accurately labeled.

BAI researchers brought the scientific method to agriculture--testing, measuring, and comparing experimental models against careful controls in a myriad of experiments. The results, over the decades, have made America a world leader in scientific agricultural information and--more importantly--have enabled farmers to take these advances and put them to work, efficiently and economically producing animals for the American consumer. Two examples: today's leaner meat-type hogs and the Beltsville turkey--a smaller bird with a high percentage of breast meat.

Today the gene-splicing techniques of the 1980's point the way toward development of new vaccines that are safer, more effective, and more economical to produce.

During 1984, the Department has asked agricultural groups throughout the United States to join in commemorating "100 years of animal health." Over 90 organizations have been invited to take part in the centennial activities. The commemoration began May 29, 1984, with special commemorative ceremonies in Washington, D.C., along with a scientific seminar on the "Second Century of Animal Health and Well-being." (Mr. L. D. Mark, APHIS, News Center, 301 436-7799).

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